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    FILE 'MEDLINE, EMBASE, BIOSIS' ENTERED AT 10:52:48 ON 07 MAY 2004
L1         20671 S HYPERTENSION AND ANIMAL MODEL
L2          1079 S L1 AND ESSENTIAL HYPERTENSION
L3             2 S L2 AND VEGF

=> s l2 and accepted
L4             5 L2 AND ACCEPTED

=> duplicate remove l4
DUPLICATE PREFERENCE IS 'EMBASE, BIOSIS'
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PROCESSING COMPLETED FOR L4
L5             5 DUPLICATE REMOVE L4 (0 DUPLICATES REMOVED)

=> d 1-5

L5   ANSWER 1 OF 5  BIOSIS  COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN   2002:42259  BIOSIS
DN   PREV200200042259
TI   Intrauterine programming of nephron number: The fetal flaw revisited.
AU   Marchand, Michael C.; Langley-Evans, Simon C. [Reprint author]
CS   Division of Nutritional Biochemistry, University of Nottingham, Sutton
     Bonington Campus, Loughborough, LE12 5RD, UK
     Simon.Langley-Evans@nottingham.ac.uk
SO   JN Journal of Nephrology, (September-October, 2001) Vol. 14, No. 5, pp.
     327-331. print.
     ISSN: 1121-8428.
DT   Article
     General Review; (Literature Review)
LA   English
ED   Entered STN: 2 Jan 2002
     Last Updated on STN: 25 Feb 2002

L5   ANSWER 2 OF 5  EMBASE  COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED.
     on STN
AN   97238915  EMBASE
DN   1997238915
TI   Diet, genetics and hypertension.
AU   Preuss H.G.
CS   Dr. H.G. Preuss, Georgetown University Medical Center, 4000 Reservoir Rd
     NW, Washington, DC 20007, United States
SO   Journal of the American College of Nutrition, (1997) 16/4 (296-305).
     Refs: 164
     ISSN: 0731-5724  CODEN: JONUDL
CY   United States
DT   Journal; General Review
FS   005     General Pathology and Pathological Anatomy
     006     Internal Medicine
     017     Public Health, Social Medicine and Epidemiology
LA   English
SL   English

L5   ANSWER 3 OF 5  EMBASE  COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED.
     on STN
AN   95362379  EMBASE
DN   1995362379
TI   Transgenic animals in the study of blood pressure regulation and
     hypertension.
AU   Thompson M.W.; Merrill D.C.; Yang G.; Robillard J.E.; Sigmund C.D.
CS   Transgenic Animal Facility, Dept. of Medicine, Univ. of Iowa College of
     Medicine,Iowa City, IA 52242, United States
SO   American Journal of Physiology - Endocrinology and Metabolism, (1995)
     269/5 32-5 (E793-E803).
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ISSN: 0193-1849 CODEN: AJPMD
CY United States
DT Journal; General Review
FS 002 Physiology
006 Internal Medicine
018 Cardiovascular Diseases and Cardiovascular Surgery
022 Human Genetics
037 Drug Literature Index
LA English
SL English

L5 ANSWER 4 OF 5 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 1992:162824 BIOSIS
DN PREV199293085149; BA93:85149
TI SPONTANEOUSLY HYPERTENSIVE AND WISTAR KYOTO RATS ARE GENETICALLY DISPARATE.
AU H'DOUBLER P B JR [Reprint author]; PETERSEN M; SHEK W; AUCHINCLOSS H; ABBOTT W M; ORKIN R W
CS VASCULAR DIV, SURG SERV, MASSACHUSETTS GENERAL HOSP, DEP SURG, HARVARD MED SCH, BOSTON, MASS 02114, USA
SO Laboratory Animal Science, (1991) Vol. 41, No. 5, pp. 471-473.
CODEN: LBASAE. ISSN: 0023-6764.
DT Article
FS BA
LA ENGLISH
ED Entered STN: 31 Mar 1992
Last Updated on STN: 31 Mar 1992

L5 ANSWER 5 OF 5 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
AN 85033735 EMBASE
DN 1985033735
TI Studies on the effect of a chemical sympathectomy on the enzyme pattern in the heart of spontaneously hypertensive rats (SHR) fed diets supplemented with different polyunsaturated fatty acids (PUFA).
AU Papies B.; Wagenknecht C.; Konig M.-L.; et al.
CS Institute of Pathological and Clinical Biochemistry, Humboldt-University, Berlin, Germany
SO Biomedica Biochimica Acta, (1984) 43/8-9 (S179-S183).
CODEN: BBIADT
CY Germany
DT Journal
FS 003 Endocrinology
018 Cardiovascular Diseases and Cardiovascular Surgery
029 Clinical Biochemistry
002 Physiology
LA English

=> d 1-5 abs

L5 ANSWER 1 OF 5 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AB A broad range of epidemiological evidence supports the hypothesis that risk of **essential hypertension**, coronary heart disease and non-insulin dependent diabetes is, in part, determined before birth. This phenomenon, termed programming, is now the subject of intensive investigation in order to determine possible underlying mechanisms. It is widely **accepted** that maternal nutritional status in pregnancy is a major programming influence upon the fetus. This review considers the hypothesis that nephron number in humans is determined by prenatal nutrition. An increasing number of human studies indicate that the developing kidney is particularly vulnerable to the adverse effects of fetal growth retarding influences. In animals, growth retarding diets or other insults which have an impact upon the development of cardiovascular

functions, also appear to impact upon nephron number. However, it is possible that **hypertension** and reduced renal reserve merely coincide and are not causally associated.

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AB It is generally **accepted** that genetics play a significant role in the pathogenesis of **hypertension**. Since **hypertension** often follows kidney transplantation, candidate genes have been sought and found in the kidneys of rats and humans. One well-recognized, inherited influence on blood pressure (Bp) occurs via abnormal renal sodium handling in vivo. Further, abnormal renal sodium handling is seen in isolated kidneys of genetically hypertensive rats. People who have a relative inability to handle a sodium lead properly, and retain it inappropriately, often develop high BP and are referred to as 'salt-sensitive'. More than half of patients diagnosed with **essential hypertension** are salt-sensitive. In contrast to the deleterious effects associated with high sodium intake, many believe that ingestion of more potassium, calcium, and magnesium may influence BP favorably. The beneficial effects of these ions work, at least in part, through an effect on sodium balance, i.e., a diuretic influence. In support of this concept, they lower BP more effectively in salt-sensitive hypertensives. Refined carbohydrates and saturated fats are also associated with salt retention and **hypertension**. Thus, dietary factors, working directly on their own and/or indirectly via effects on genetic mechanisms, may alter BP favorably or unfavorably.

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AB It is generally **accepted** that the etiology of **essential hypertension** is due to a complex interplay of genetic and environmental factors. A great deal of research effort over the past ten years has been focused on the identification of genes the variants of which predispose individuals to high blood pressure. Consequently, transgenic and knockout animals have become important research tools, providing experimental systems in which defined genetic manipulations can be introduced on uniform genetic backgrounds while minimizing environmental variation. These animal models have provided the means by which candidate genes thought to be involved in blood pressure regulation have been studied. Furthermore, these models can be used to test the significance of genes and gene variants identified via genome-wide searches as potential causes of **hypertension**. The purpose of this review is to provide a brief discussion of transgenic and knockout methodology and its application to study the genetic basis of **hypertension**.

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AB Spontaneously hypertensive rats (SHR) are one of the most common animal models used to study **essential hypertension** in humans. Because SHR and normotensive Wistar Kyoto (WKY) rats were both established from the same parenteral, normotensive Wistar stock, WKY animals have been used almost exclusively as control animals in studies of SHR. Recently, the suitability of WKY rats as normotensive controls for SHR has been challenged. To establish whether or not SHR and WKY rats share the same immunologic backgrounds, we initially performed a series of skin grafting experiments on these animals. In all cases, grafts of SHR donor skin to WKY recipients and of WKY donor skin to SHR recipients resulted in complete rejection within 7 to 10 days. In addition, grafts of WKY donor skin to other WKY recipients resulted in graft rejection. By contrast, skin grafts between SHRs were always **accepted**. To further characterize the genetic distinctions between SHR and WKY rats, allelic profiles based on a series of immunologic and biochemical markers were established for each strain. These findings clearly establish that SHR and WKY rats differ at the major histocompatibility complex, in specific

blood group antigens, and in a panel of isozymic markers. Moreover, whereas SHRs have the same genetic profiles irrespective of source, some colonies of WKY rats are outbred, as judged by their variant allelic profiles.

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AB Spontaneously hypertensive rats are generally **accepted** as a suitable model for **essential hypertension** in man at least with regard to some etiological and pathogenetic aspects. In these animals the genetically determined **hypertension** manifests itself at an age of about 8-10 weeks. The cause of blood pressure elevation is hitherto not fully clear. It is suggested that an increased activity of the sympathetic nervous system which has been described in early stages of spontaneous **hypertension** in rats serves as an initiating mechanism for **hypertension**. This elevated sympathetic activity might at least be partly related to disturbances in the metabolism of PUFA and prostaglandins taking into account the known negative feedback of E prostaglandins on transmitter release (1). The influence of LA supplemented diet on catecholamines and dopamine β-hydroxylase in the adrenals of SHR (2) as well as the blood pressure lowering effect of prenatal feeding PUFA rich diets (3) are in agreement with this suggestion. In a previous study we have shown that not only the blood pressure of SHR but also **hypertension**-linked complications reflected by alterations of the enzyme pattern in heart and liver can be beneficially influenced by prenatal application of PUFA enriched diets. It was the purpose of the present study to investigate: 1. the influence of postnatal feeding PUFA rich and PUFA deficient diets on blood pressure and myocardial enzyme pattern, 2. the effectiveness of different PUFA in influencing blood pressure and enzyme pattern, 3. the effect of chemical sympathectomy in PUFA rich and PUFA deficient SHR.

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